THE PRESENT STATUS OF CLINICAL ELECTROENCEPHALOGRAPHY*

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THE clinical investigator has found in electroencephalography a powerful tool with which to attack some of the most baffling of the problems of nervous and mental disease. It is the purpose of the present report to summarize certain recent advances and to indicate what use can now be made of the electroencephalogram in practical clinical studies.

Before proceeding to this task, honor should be paid to those who pioneered. It was, of course, Hans Berger who pointed the way.[1] He showed that the electroencephalogram is surprisingly easy to record, but more importantly that it changes with age, with attention and with states of impaired consciousness. Credit should go to Fischer[2] who showed that abnormal cortical potentials appear during convulsions, and to Tönnies who demonstrated the practicability of ink-writing electroencephalographs,[3] and to Albert Grass, who provided superb equipment for electroencephalographers throughout the world.[4] The contributions of Kornmüller,[5] Grey Walter,[6] Adrian,[7] Bremer,[8] Jasper[9] and others[10] gave basic information and orientation to all early investigators.

In the past 30 years practically every normal and abnormal state of central nervous function in man and animals has been surveyed electroencephalographically. Tens of thousands of men, women and children have been studied with multi-channel equipment. Thousands of miles of records have been inspected and correlated with various clinical conditions. This work has placed electroencephalographic diagnosis on a solid statistical basis, but it has done more than that; it has made the electroencephalographer familiar with his instrument and his material so that he can look through the electroencephalogram at the

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brain.* In this respect he is like the roentgenologist who looks not at the grains of emulsion on the X-ray film, but at the picture of body structures. The electroencephalographer sees the 400 feet of paper with six or eight wiggly lines on it that forms the electroencephalogram, not in terms of its primary aspects but in terms of its neurophysiological and clinical correlates. Five sets of data have shown important correlations with the electroencephalogram: 1) brain metabolism; 2) age; 3) the level of consciousness; 4) the clinical symptomatology of epilepsy and related brain disorder (trauma, encephalitis, vascular disease, etc.); 5) the pharmacological action of stimulants, sedative and anti-epileptic substances.

The electroencephalogram gives evidence of a type of activity which must be described in general terms because, though it is found more highly developed in nerve cells than in the other tissues, it is nevertheless characteristic of protoplasm in general. It has been termed irritability; the tendency to release stored energy after thermal, chemical, mechanical or electrical stimulation. Voltage production is one form of energy release.

A tendency to rhythmic electrical activity is inherent in all neurone aggregates (ganglia, cord and brain). The cortex shows electrical pulsations with a voltage of approximately 5-500 microvolts and a frequency of 1-50 cycles per second. The primary sources of energy for the fluctuating voltage are glucose and oxygen. To use a mechanical analogy, these two substances are the mainspring of the oscillator, but the escapement or control of energy release lies elsewhere: in CO_2 tension[11] and in as yet unknown enzymes and neuronal mechanisms. Obviously any tissue which has as a major characteristic the release of energy must be provided also with mechanisms for storing energy and regulating its release. Although these mechanisms are not yet understood their operation is manifest in the electroencephalogram, for here one sees energy (derived primarily from the blood stream) transferred to the nervous

* Persons professionally interested in electroencephalography are now organized in many countries. The names, secretaries, and number of members of some of these societies are as follows:

English—The Electroencephalographic Society, Great Britain, W. Grey Walter, M.D., Secretary, Burden Neurological Institute, Bristol, England. 72 members.

French—Société D'EEG et des Sciences Connexes de Langue Française, Henri Gastaut, M.D., Secretary, 149 Promenade de la Corniche, Marseille, France. 50 members.

Italian—Italian Electroencephalographic Society, G. Moruzzi, M.D., Secretary, University of Pisa, Pisa, Italy. 30 members.

American—American Electroencephalographic Society, Dr. Robert Schwab, Secretary, Massachusetts General Hospital, Boston 14, Massachusetts. 107 members.

These societies have united to publish "Electroencephalography and Clinical Neurophysiology, an International Journal." Dr. Herbert Jasper, Neurological Institute, Montreal, Canada, is Editor-in-Chief.

system in finely graded quantities and channeled through neuronal pathways in a precisely regulated manner. This regulation of energy release is of extreme importance for proper function. Modulation or control of energy release in the nervous system is of two types: In the nerve fibers where conduction is the primary function, amplitude is held relatively constant and frequency is modulated (as is conventional in radio communication whenever interference must be kept to a minimum), but in synaptic areas (near the cell body) where interference, interaction and mixing are essential to normal function, amplitude modulation is combined with frequency modulation.

If more than the usual amount of energy is released, as evidenced by increased voltage production per unit time, clinical signs of hyperactivity appear. If less than the usual amount of energy is released, as evidenced by a decrease in the voltage production per unit time, clinical signs of depression of neuronal function appear. The specific symptoms that develop depend on the function subserved by the structures involved in the abnormal energy release. Thus it can be said that the general character of the dysfunction (hyper or hypo) and its time course are electro-chemically determined, but the specific symptom is anatomically determined.

Electroencephalographic patterns have their simplest form and the electrical sign (negativity or positivity) is most easily interpreted when voltage readings from points on the scalp (or some part of the brain) are referred to a relatively inactive reference point (usually the ear lobe). This has been called "monopolar recording." The term is inappropriate, however, because recording with one electrode is impossible. The term "bi-polar" has been applied to recordings from serial pairs of electrodes arranged in the form of a triangle or a line, the activity of each pair being referred back or forward to the next pair. The final description of voltage conditions in the brain will be the same whether activity is recorded with a common reference or with a series of references.

When the electrical activity of the brain is recorded between two electrodes A and B on the head, the possibility arises that both are active and that the resultant tracing is the algebraic sum of the two activities. "Bi-polar" recording tries to subtract from the sum of the activity at A and B other sums obtained by using A and B in conjunction with other points, thus arriving at an evaluation of the voltage con-

ditions at A and B. When a common reference is used the "stigmatic" electrodes are placed at A and B and any difference between the activity recorded is attributable to differences in voltage at A and B, the points in question; thus also for activity recorded from all other points to the common reference. In some cases, however, universal activity may be registered which may be due to activity at the reference point. This may be checked by moving to another reference. However, the same difficulty may arise with the new reference. By removing the reference point further away from the presumed voltage source, shunting is obtained which will tend to reduce the pick-up of activity. For most purposes the ear lobes are sufficiently removed from the brain to serve as satisfactory reference points. However, the anterior temporal spike focus of psychomotor epilepsy, which will be discussed later, usually spreads to the ear, and slow activity is also commonly recorded from the ear in cases with a tumor of the temporal lobe. The opposite ear may be used as reference in such cases or the vertex or occipital region may provide a satisfactory "quiet" reference, or recourse may be had to electrodes on the nose or on the chin. In order to get well away from the brain it may be desirable to place electrodes on the chest and use these as a reference.[12] The complication introduced by the electocardiogram can be avoided if electrodes are placed symmetrically on opposite sides of the heart and interconnected through a potentiometer. Rather than pick up the full voltage of the heart it is desirable to keep somewhat away from it, and the positions ordinarily chosen for chest electrodes are the spine of the first thoracic vertebra and the right sterno-clavicular junction.

From animal experiments[13] and from recordings in the depths of the human brain[14] a negative voltage (referred to a relatively inactive reference) indicates that the electrode is in or on the discharging area. A positive discharge indicates that the electrode is beyond the discharging area.

Excessive thermal, mechanical, chemical or electrical stimulation slows, decreases and finally abolishes cortical voltage production.[15] Thus, flattening of the electroencephalogram and disappearance of the brain waves can be considered an extreme form of slowing.* Depression

* A decrease in amplitude to the point of flattening is rarely encountered clinically. It is an extreme change which carries with it an immediate danger of death. A depression of amplitude resulting in an asymmetry of voltage production from homologous areas in the two hemispheres is a sign of minimal damage and has some clinical localizing value.

of voltage production (slowing or flattening) is a *primary* reaction to injury. In some cases, after mild depression of voltage production or during the phase of recovery from severe depression, a secondary disturbance develops; it consists of paroxysmal *excessive* voltage production. As indicated by the sudden failure of cortical function when the blood supply is cut off, the normal energy consumption of the brain is almost identical with the rate at which it is supplied. Therefore, abnormally great release of energy in the cortex is quickly limited by failure of energy supplies and followed by diminution or absence of voltage production. Excessive voltage production tends to spread, however, and to drive neighboring and connected neurones into similar activity. The greater the excess of voltage the greater the spread and the less precisely the voltage is held within the usual neuronal channels. This reaction of paroxysmal excessive voltage production is an irritative reaction to injury; it constitutes the pathological, physiological basis of epilepsy.

From the electroencephalographic point of view, epilepsy is a disorder of rate regulation which manifests itself as a tendency to abnormally violent and extensive discharge, i.e., as a failure to provide proper spacial and temporal limitation of voltage production. This failure of rate regulation is produced by the same agents which produce normal alterations of rate regulation, mechanical, thermal, electrical or chemical stimulation, but these must surpass certain physiological limits of intensity and duration. Epilepsy can be considered a "tumor of function;" a distortion of one of the chief normal functions of neurone aggregates (i.e., voltage production). Just as a true tumor can be considered a disturbance of temporal and spacial regulation of the rate of reproduction (a feebly represented function in neurones) so an epileptic seizure can be regarded as a failure of the temporal and spacial regulation of energy release. In this view epilepsy is not a disease, but a type of dysfunction. It can have no single etiology and will have as varied a symptomatology as the nervous system has specific receptor, effector and integrator functions. It will certainly be caused by many types of injury and almost any type of injury will be found in some cases to be imitated by a genetic defect.

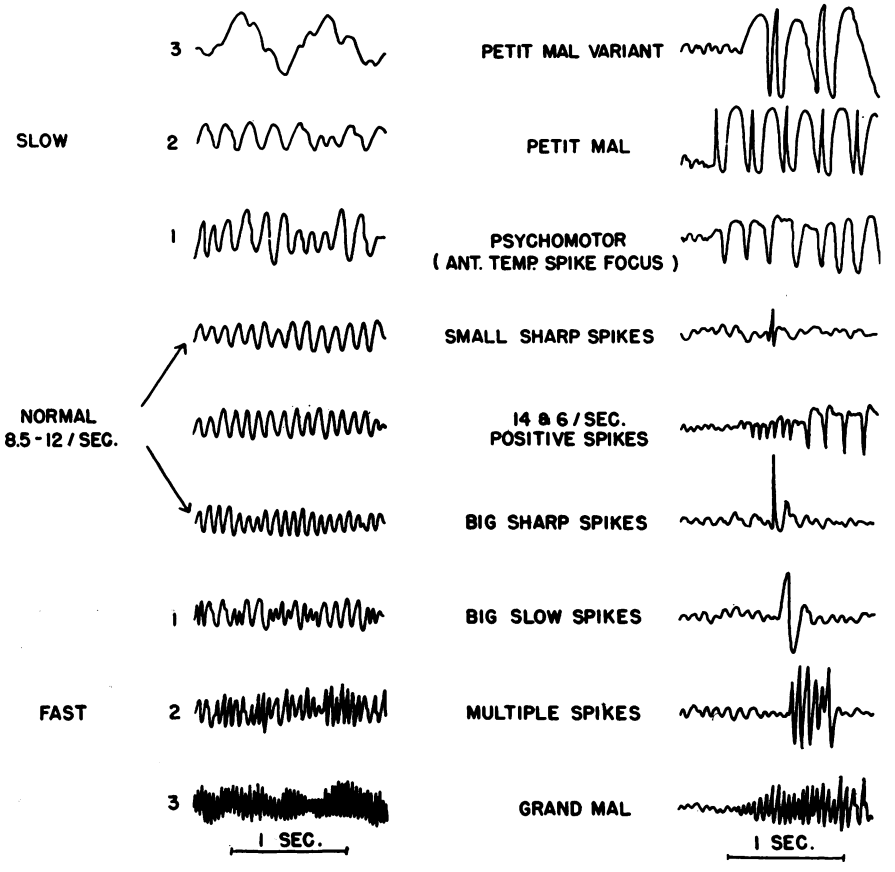
In epilepsy three general types of abnormal voltage production are encountered. They are referred to as seizure patterns.[16] The first is a discharge of unusually fast waves of increasing amplitude followed by interrupted fast waves and later by slow activity. This is the classical

tonic-clonic or grand mal type of seizure discharge (Fig. 1). If the entire brain is involved maximal neuronal activity becomes manifest in generalized muscular contractions and complete loss of consciousness. From the point of view of energy consumption it represents a supermaximal discharge which cuts deeply into the energy reserves and requires a relatively long period of recovery (stupor) before normal voltage production returns. The second type of discharge is the spike-and-wave pattern which appears in its most perfect form as the three-per-second wave-and-spike of petit mal epilepsy (Fig. 1). In this type of discharge each spike of excessive voltage production is followed by a third-of-a-second of stupor (the slow wave) which apparently allows for recovery of function and the possibility of another discharge. However, as the pattern continues the spike usually tends to drop in voltage and the frequency of the repetitive pattern tends to slow from an initial approximately four-per-second to a final two-per-second rhythm. This type of discharge is rarely followed by stupor; it is a relatively conservative paroxysmal dysrhythmia, and is less disorganizing to central nervous function than the grand mal type of discharge. It is associated with three-per-second clonic movements of the head and eyes, posture is usually maintained, and consciousness is commonly diminished but not completely lost. The third type of seizure pattern consists of high-voltage six-per-second and square or saw-toothed four-per-second waves. This type of discharge is referred to as a psychomotor discharge (Fig. 1). It is associated with mental confusion and poorly coördinated but apparently purposeful movements. It is rarely followed by stupor. The psychomotor pattern is the one which is the most nearly normal of the three chief types of seizure discharge. Further study has shown that it is associated with a focus of negative single spike-seizure activity in the anterior temporal region.[17]

A single spike (Fig. 1) is the minimal epileptic discharge; it is commonly asymptomatic, and commonly focal. Spike foci can occur anywhere in the cortex, but they are particularly common in the anterior temporal region. A spike focus in the anterior temporal region has been called a psychomotor type of focus because it is usually associated with psychomotor seizures (trance-like attacks and confusional episodes). The extreme commonness of a focus of spike-seizure activity in the anterior temporal region suggests that this is the most vulnerable of all cortical areas to the irritative type of injury which underlies epilepsy.[18]

FIGURE I

TYPES OF NORMAL AND ABNORMAL ELECTROENCEPHALOGRAM



In the left column are shown the non-paroxysmal patterns; above are those that are abnormally slow for adults, and below, those that are abnormally fast. In the middle are patterns which fall in the 8½ to 12 per second range which is normal for adults. The scale of abnormality reads from one to three, with one being slightly abnormal; two, very abnormal, and three, exceedingly abnormal. This scale, however, must be adjusted for age. The paroxysmal patterns shown in the righthand column are given descriptive names or are named after the clinical type of seizure with which they are most commonly associated. The pattern referred to as "psychomotor" appears as a generalized disorder over the entire cortex during a clinical seizure of the psychomotor type. In seizure-free intervals, particularly during sleep, the psychomotor disorder appears as a focus of negative spike seizure potentials in the anterior temporal region. The 14 and 6 per second positive spikes are in reality two patterns which can appear independently as 14 or 6 per second positive spikes.

It is not enough to determine that the electroencephalogram shows seizure discharges or epileptic waves. The type of wave is important for diagnosis and prognosis. Seizure discharges of the grand mal type are benefited by Dilantin,* phenobarbital and Mesantoin,* whereas the three-per-second spike-and-wave of petit mal responds to Tridione* and Paradione.* Seizures of the psychomotor type are commonly resistant to medication.

In the waking state, with electrodes fastened to the scalp, the activity of the underlying cortex is recorded. During sleep deeper centers take over the regulation of cortical activity. Voltage production in sleep is generally conservative. i.e., slower than the normal waking activity, but with deeper sleep slower and slower potentials appear.[19] The patterns at different levels of sleep are so different that it seems reasonable to suppose that the pacemaking drive or control of cortical frequency is progressively shifted to slower and *lower* centers. Sleep projects some types of subcortical disorder onto the cortex where they can be seen by the electroencephalographer with surface electrodes.[20] It potentiates all types of seizure activity; it largely eliminates age difference; it makes possible recordings on uncoöperative patients. It more than doubles the total clinical value of electroencephalography.

Over 50 per cent of patients with psychomotor epilepsy (i.e., confusional episodes and an anterior temporal spike focus) have personality disorders;[18] in half of these the personality disturbance is so severe that it can be classified as a psychosis. The psychiatric disorder is independent of the epileptic component; it is likely to become intensified when the psychomotor seizures are controlled with medication.[22] Anterior temporal lobectomy, which has been tried by Dr. Percival Bailey, has resulted in improvement of both the psychiatric and epileptic disorders in some cases.[23] The anatomical structures involved in the epileptic and psychiatric symptomatology of psychomotor epilepsy are the same, but the physiological basis of these two components are antithetic. This is probably the same antithesis which is employed for therapeutic purposes in convulsive shock therapy.

Another type of paroxysmal discharge consists of 14 and 6 per second positive spikes which appear in light sleep.[24] It is believed that because of the positive electrical sign of these discharges they come from

* These are trade names for 5,5 diphenylhydantoin; 3 methyl 5, 5-phenylethyl hydantoin; 3,5,5-trimethyloxazolidine 2,4-dione; and 3,5-dimethyl-5 ethyloxazolidine 2,4-dione.

the depths of the brain. The frequency of 14 and 6 per second suggests that they arise in the thalamus where potentials of this general frequency have been localized by Morison et al[25] in the general region of the internal medullary lamina. Such discharges are usually associated with a history of "fainting spells" and a variety of symptoms suggesting thalamic disturbances.

A report in preparation[26] will deal in detail with exceedingly fast non-paroxysmal activity (35-40 per second) (Fig. 1, F-3) which appears best in the precentral regions during light sleep. This, like very fast activity (Fig. 1, F-2), correlates with epilepsy and related organic brain disorder, but it is commonly associated with personality disturbances and is particularly common in post-traumatic psychosis.

By regarding focal evidence of primary and secondary reactions to injury the informed electroencephalographer with adequate equipment is able in many instances to localize lesions which are not apparent from neurologic or roentgenographic examination. Thus, the modern neurosurgeon or neurologist finds the electroencephalograph indispensable. He will not be surprised or too disappointed by cases in which a huge atrophy or widespread demyelination shows little or nothing in the electroencephalogram, for destruction of neural elements does not show well in the electroencephalogram; absence of activity is by no means as evident as disordered activity. An inflammatory process, like acute encephalitis, tends to give maximal electroencephalographic findings even in cases where there are minimal clinical signs, and this helps to distinguish it from a more chronic disorder, for example, multiple sclerosis where the electroencephalographic findings are usually slight compared with the clinical signs. Likewise, a small metastatic carcinoma in the cerebral cortex, because of its rapid growth, usually produces more electroencephalographic disorder than a huge but slow-growing meningioma. If electroencephalography is to be used successfully by the clinician he must familiarize himself with it and realize that the electroencephalograph shows functional rather than structural disorder. The severest electroencephalographic disturbances, for example, convulsions and stupor, are not usually associated with demonstrable structural change. A violently discharging epileptic focus, when identified at operation by direct recording from the cortex, and when ablated by the neurosurgeon, commonly appears normal to microscopic examination.[27]

Although electroencephalography is of value as a supplementary aid

to classical neurology and neurosurgery, which subjects deal with gross or microscopic evidence of structural changes (pathological anatomy), it does something more, something new. Although it does not correlate well with anatomical changes and signs of structural deficits which are essentially irreversible, it does correlate with pathological-physiologic changes which are reversible and amenable to medical and surgical therapy. It opens a new field of dynamic neurology and physiological psychiatry. Hans Berger would be happy to see how his brain child has prospered.

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